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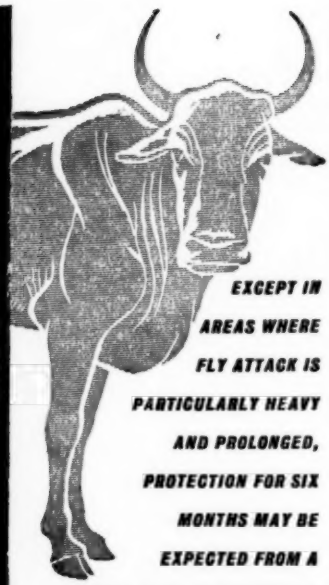

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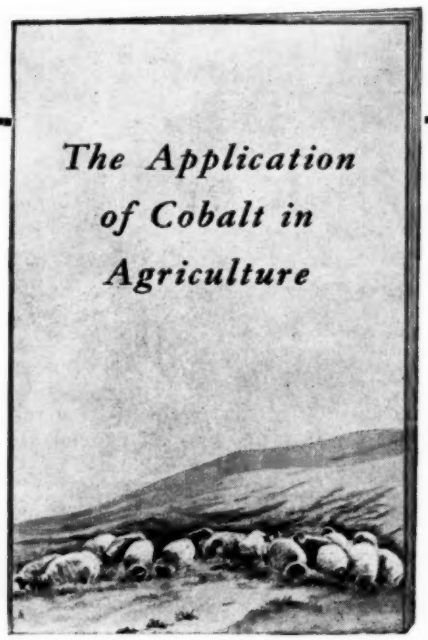

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The Signs and Stages of Anaesthesia in Domestic Animals

BY

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University of Glasgow Veterinary School

SUMMARY.—All anaesthetic drugs are highly potent, toxic agents and since one of the principal objectives of anaesthesia is the achievement of conditions of maximal safety, anaesthetics should be administered only by trained personnel who are familiar with the signs and stages of anaesthesia, the individual drug differences, and the variations of reaction that occurs in the different species. If this precaution is observed anaesthetic fatalities can be reduced to a minimum.

Guedel's classification of the signs and stages of anaesthesia has been employed with minor modifications, as a suitable basis for determining the depth of anaesthesia in animals.

The action of anaesthetic drugs and their effects on vital functions is discussed. Major stress is laid upon the effects of anaesthetics on respiration, since this is the most important factor employed in the assessment of the depth of anaesthesia. It is a primary duty of the anaesthetist to make certain that excessive respiratory depression, or respiratory collapse does not occur as this may lead to death.

The other signs which may be used as an aid in assessing the level of anaesthesia; eye signs, muscle tone, etc., are also discussed and their significance stated.

The influence of oxygen want and of variations in carbon dioxide tensions are discussed and the main actions of the principal volatile anaesthetic agents given.

IN recent years there has been a great increase in the scope of surgery in both human and veterinary fields. This is due in no small measure to the fact that modern anaesthetic methods allow the production of suitable conditions for various types of interference and at the same time give a high degree of safety.

In order, however, that general anaesthesia should be as safe as possible, it is necessary that the anaesthetist should have a clear understanding of the significance of the various signs by which the level of anaesthesia may be judged. Although in recent years there have been a number of publications on anaesthesia in the veterinary literature (Hall, 1955; Hall & Weaver, 1954; Longley, 1950; Joshua, 1950), an adequate description of the signs by which the level of anaesthesia in animals can be judged has not, to the authors' knowledge, appeared to date.

The introduction of barbiturates by Wright (1934a, b) and their use intravenously was one of the factors which contributed to the ease with which animal surgery could be carried out and was responsible for a very considerable increase in general surgical work. More recently it has become recognised that while barbiturates serve a very useful purpose, anaesthesia with volatile agents is attended with a greater degree of delicacy of control, *i.e.* a higher degree of safety. The various features of volatile anaesthetics which render them more valuable include rapid recovery on withdrawal; the ability of the anaesthetist to adjust the level to the desired depth; the ease with which resuscitation can be effected following respiratory collapse.

Under most systems of administration the depth of anaesthesia can vary with considerable rapidity and this fact makes it essential that the anaesthetist should be familiar with all the signs of anaesthesia so that instant recognition of alteration in the anaesthetic level is possible.

The object of this communication is to set out clearly the details of the signs by which the level of anaesthesia may be judged.

Guedel (1937) devised a classification for the various stages of ether anaesthesia in man and this

proved to be a very valuable method of assessment of depth of anaesthesia for all volatile anaesthetic agents and to a slightly more limited degree for intravenous agents. Although this classification was devised for use in human anaesthesia it has been used in this hospital for a considerable number of years and has proved eminently suitable as a basis for anaesthetic administration in animals.

Classification of Stages of Anaesthesia According to Guedel

Stage I.—Analgesia. This stage lasts from the beginning of induction to loss of consciousness with increasing loss of pain sensitivity. In animals the application of an irritant vapour is frequently attended by excitement and active resentment and these features necessitate the use of some means of restraint. The degree of excitement may be reduced by pre-anaesthetic sedation.

Stage II.—Delirium. This stage lasts from the onset of unconsciousness to the onset of automatic respiration. The influence of the higher centres is removed and causes loss of self-control.

This stage is evident in both animals and man and is associated with struggling, breath holding, and possibly vomiting. These features may appear during induction or recovery, *i.e.* during deepening or lightening anaesthesia.

The use of intravenous barbiturates for induction will frequently cause a sufficiently rapid transition through Stages I and II so that their recognition is difficult.

Stage III.—Surgical anaesthesia. This stage lasts from the onset of automatic breathing to apnoea (cessation of respiration) from respiratory paralysis. This is the stage of progressive muscular depression and for convenience it is separated into 4 planes. The detailed signs by which these planes may be recognised are of paramount importance and will be dealt with in detail later in the paper.

Stage IV.—Respiratory arrest. This stage lasts from the onset of complete respiratory paralysis to death from cardiac failure. All reflex activity is lost and the pupil widely dilated. Death in the absence of artificial respiration results from the inability of the myocardium to withstand prolonged anoxia. For this reason artificial respiration should be instituted with minimal delay so as to maintain oxygenation and prevent CO_2 build-up.

Main Action of Anaesthetic Drugs

Anaesthetic drugs all act by limiting the oxygen uptake of tissues. The effect on an individual tissue is proportional to its normal oxygen requirement. Since the oxygen requirement of nervous tissues is disproportionately high, these tissues are the first to be affected by anaesthetic drugs. Unconsciousness, abolition of reflexes, muscular atony, and respiratory paralysis are due to depression of the cerebral cortices, the mid-brain, the spinal cord, and the medulla respectively.

A constant action of any anaesthetic agent is its effect on the respiratory centre which is always depressed. This has the effect of lowering the sensitivity of the centre to its physiological stimulant—carbon dioxide, and can be referred to as an increase in the respiratory threshold. The changes which take place in respiration are the result of this effect on the respiratory centre.

In normal respiration the movements of the two components, the chest wall, and the diaphragm, are synchronous; the chest wall expanding at the same time as the diaphragm contracts, on inspiration. The diaphragm and intercostal muscles relax at the same time and expiration is due to the normal elastic recoil of the thoracic cage and the lungs, *i.e.* expiration is passive. (In this connexion it should be noted that during positive pressure respiration in a closed circuit, the rebreathing bag should be completely released during expiration in order to permit this elastic recoil to produce maximal exhalation.) After each expiration, when the animal is breathing normally, there is a short period of respiratory inactivity—the expiratory pause. Thus the sequence in normal respiration is: inspiration, expiration, pause——, inspiration, expiration, pause——. (See Fig. 1.)

The Signs of Anaesthesia

I. Changes in Respiration

In Stage I respiratory volume gradually increases due to stimulation of the respiratory centre (especially with ether).

In Stage II breathing becomes irregular and is often jerky, but the "force" of expiration is normal.

Stage III

Plane 1. The beginning of Stage III is indicated by the sudden onset of regular automatic respiration of normal physiological character. The depth of breathing depends on the relationship between the respiratory threshold and the amount of stimulant—CO_2—in the blood. Presedation depresses, while surgical trauma stimulates respiration in Planes 1, 2, and 3.

Plane 2. There is little difference in the respiration in Plane 2, the division between the first two planes being judged by other signs.

Plane 3. In this plane the intercostal muscles are progressively paralysed. This results in a degree of "quietening" of respiration at first and a gradual decrease in tidal volume as deeper levels are reached. The intercostal paralysis is progressive and chest movements tend to occur slightly later than diaphragmatic movements—intercostal lag. The respiratory rate may rise to compensate for the diminished depth of respiration.) At the lower border of the plane respiration is entirely abdominal in character and becomes somewhat gasping. This may cause annoyance to the surgeon by producing excessive movement of abdominal viscera. This must not be mistaken for very light anaesthesia, the appropriate action being to lighten, not deepen, the level of anaesthesia.

Plane 4. The thorax is entirely passive in Plane 4. Thus the normal movements of the chest wall become reversed. On inspiration the chest at first collapses due to the reduction in intra-thoracic pressure, while on expiration, the increase in intra-thoracic pressure

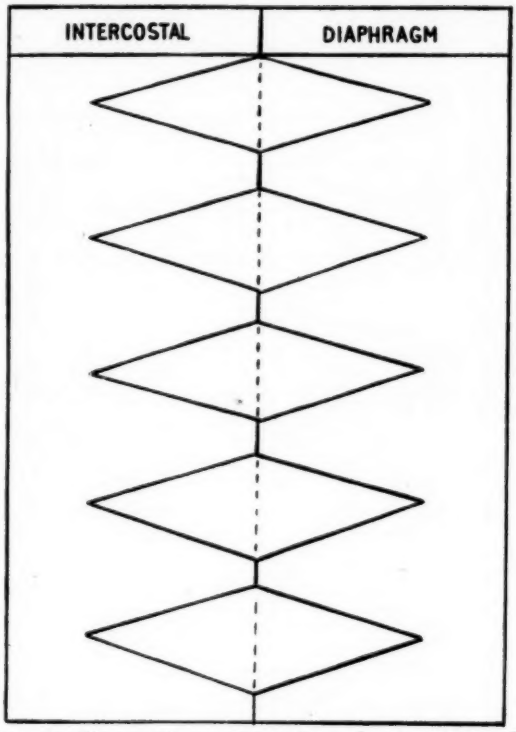

FIG. 1.—Diagramatic representation of movements of respiratory muscles.

The left half represents the thoracic component and the right half the diaphragmatic component.

The central line represents the state of relaxation of the respiratory muscles.

The solid vertical line between the diamonds represents the expiratory pause.

Contraction of intercostal muscle and diaphragm occur simultaneously on expiration.

The regularity of the rhythm, as illustrated, is more noticeable in Stage III, Plane 1, anaesthesia, than during conscious respiration, since it is then freed from interference due to extraneous stimuli.

The various features illustrated may be studied by observing:—

(1) The rebreathing bag; when the volume of gas inhaled and exhaled may be noted and the rhythm, with its expiratory pause, studied.

(2) The anaesthetised animal, when note should be made of both thoracic and abdominal movements.

(*Note.*—With increasing depth of anaesthesia these components show variations from the features noticeable in Plane 1.)

consequent on relaxation of the diaphragm causes the chest wall to expand and then gradually sink to the normal level. At this level in Plane 4 it can be seen that the movements of the chest and abdomen assume a "see-saw" movement. On diaphragmatic contraction (inspiration) the abdominal wall rises while the chest wall is retracted and on diaphragmatic relaxation (expiration) the abdomen sinks while the thorax expands.

Tidal volume is reduced since this abnormal thoracic movement hinders gas exchange and this is evinced by diminution in movements of the rebreathing bag. The force of expiration is also reduced since the elastic recoil of the thoracic wall no longer assists expiration. As Plane 4 progresses the diaphragmatic movements become smaller and smaller, *i.e.* respiration becomes shallower, until paralysis occurs. Decreased tidal exchange causes oxygen want and CO_2 excess. If these latter occur, short, infrequent respirations may ensue until apnoea from over-anaesthetisation is established. At the lower levels of Plane 4 movements of the accessory muscles of respiration may be seen especially in the dog. These cause a gasping movement of the mouth which is distinguishable from the mouth movements which occur during lightening anaesthesia, when the animal is about to pass from Stage III, Plane 1, into Stage II, by a very small tidal volume that is present. Thus it can be seen that with increasing depths of anaesthesia the respiratory centre threshold is gradually raised until it is finally completely insensitive to carbon dioxide stimulation and this is reflected in the gradual transition from normal respiration to complete respiratory collapse.

II. Effect on Circulation

The effects on circulation, as judged by the pulse, are somewhat similar. During Stages I and II, the pulse is strong and fast. As Stage III supervenes it assumes a more even rhythm, slightly faster than normal. As the depth of anaesthesia increases, depression of the vasomotor centre, coupled with relaxation of the skeletal muscles, causes a drop in blood pressure which can be appreciated in the pulse. In dogs, the labial artery, running forward on the buccal surface of the lip from the level of the 3rd upper premolar, is an extremely good guide as to the effects of anaesthesia and operative interference on the circulation. It can be assumed that, if the labial pulse cannot be appreciated, the drop in blood pressure has reached serious levels and steps should be taken immediately to improve the circulation.

III. Effect on Skeletal Muscle

Muscle tone is increased in Stage II. Surgical anaesthesia causes muscular relaxation, the different agents varying in their efficacy in this respect and the different muscle masses varying in their susceptibility. Thus the smaller skeletal muscles are relaxed in Plane 1; relaxation proceeds through successively large groups until at the bottom of Plane 3 there is complete skeletal muscular atony.

The above 3 systems are affected in the same way in all animals. The other reflexes used in estimating the level of anaesthesia vary to some extent from species to species. The levels at which paralysis of these reflexes occurs will now be discussed in detail.

IV. Eye Signs

1. *Pupil Size and Photomotor Reflex (Constriction of the Pupil on Exposure to Light)*

The photomotor reflex is abolished in Stage III, Plane 1. Thereafter the pupil becomes constricted and, as the level of anaesthesia deepens, gradually dilates again. In Stage IV the pupil is widely

dilated and does not react to light. With some agents constriction persists to the bottom of Plane 3, *e.g.* barbiturates, cyclopropane.

2. *Palpebral Reflex (Twitching of the Eyelids when the Inner Canthus is Touched)*

This reflex is abolished at the end of Plane 1 in all animals except the cat in which it is unreliable possibly due to the fact that it may be difficult to elicit.

3. *Corneal and Conjunctival Reflexes (Closure of the Eyelids on Touching the Cornea or Conjunctiva)*

These reflexes show some degree of individual variation but in general they persist only slightly longer than the palpebral reflex. Since damage to the eye may occur following repeated testing for these reflexes the authors consider that it is advisable to rely mainly on the palpebral reflex. In the horse the corneal reflex may persist to the level of respiratory failure (Wright, 1952).

4. *Eyeball Position and Movements*

In the horse and cow nystagmus occurs to the end of Plane 1. Thereafter the eyelids close until very deep anaesthesia is reached (Plane 4). In the dog, the eyeball frequently rotates downwards in Planes 2 and 3, especially when cyclopropane is being administered.

5. *Lacrimal Secretions*

These are normal in Stages I and II and are maintained in all species until the lower end of Plane 3.

In ruminants lacrimation is excessive in Planes 1 and 2 and persists rather longer than in other species.

When lower Plane 3 or Plane 4 is reached the corneal surface loses its normal sheen and appears glazed.

It is important to note that as the level of anaesthesia lightens there is delay in re-establishment of these reflexes. The animal may reach the upper end of Plane 1 anaesthesia before all the eye reflexes return.

V. Other Signs

1. *Retching and vomiting reflexes* are abolished in Plane 1.

2. *The swallowing reflex* is abolished early in Plane 1 in all animals except the cat in which it persists into early Plane 2.

3. *Laryngeal reflex (Coughing and adduction of the vocal cords are produced by laryngeal stimulation).* This reflex disappears in Plane 1 in the dog, cow, and horse. In the cat it persists till the deeper levels of Plane 2 and if stimulated may cause laryngeal stridor which can prove fatal.

Laryngeal spasm may occur due to intra-abdominal stimulation, *e.g.* traction on mesentery, mesovarium, etc., even in deep anaesthesia.

4. *Salivation.* In all non-ruminants, salivation is abolished in Plane 1. Return of salivation can be used as an indication of lightening anaesthesia.

In ruminants the production of saliva is markedly decreased but persists to the bottom of Plane 3.

5. *Anal constriction on tapping.* This is used as a guide in the horse, in which it persists to the middle of Plane 3.

6. *Pedal reflexes (flexion of the leg on painful stimulation).* These are abolished at the bottom of Plane 1, except in the pig in which they persist until deeper levels have been reached.

7. *Shivering and trembling.* The heat-control centre is depressed very early in Stage III so that shivering only occurs in Stage II and at the top of Stage III, Plane 1.

In pigs, however, muscular tremors occur during deep anaesthesia and these must not be confused with shivering.

8. *Tongue and jaw movements.* Curling of the tongue and jaw movements are abolished in Plane 1.

On lightening anaesthesia these movements may be seen concurrent with alterations in respiratory character and may be evident before return of the eye signs.

A graphic presentation of the stages and planes of anaesthesia is given below (Fig. 2 overleaf).

Oxgyen and Carbon Dioxide in Anaesthesia

In closed-circuit anaesthesia the atmosphere the animal is breathing is artificial and so care must be taken that neither oxygen lack nor carbon dioxide excess is allowed to develop. Each of these conditions produces recognisable signs, for which the anaesthetist should keep a careful watch.

Anoxia may develop due to reduced tidal exchange or too low a concentration of oxygen in the mixture administered.

Initially, in anoxia, the pulse and respiratory movements speed up in an effort to compensate. Later, as depression of the medulla progresses, respiration becomes irregular and gasping in character. Cardiac irritability is increased and ventricular fibrillation may occur, especially if adrenaline is administered. Sometimes twitching of the muscles is seen and some cyanosis develops. Anoxia of more than 4 minutes' duration may produce transient or permanent effects, *e.g.* blindness, while if the anoxia continues for 8 minutes, death results.

The level of carbon dioxide in the blood is the main factor in the control of respiration. Thus if the level of CO_2 is raised the respirations become deeper and more rapid. At the same time the vasomotor centre is stimulated, the blood pressure being raised and the pulse rate increased. As the level of carbon dioxide rises still farther the vital centres become depressed and toxic effects may be seen. Breathing first becomes deep and laboured and later shallow and gasping in character. The vasomotor centre is depressed and the blood pressure falls while the pulse rate becomes slow.

Toxic levels of carbon dioxide in the blood will produce simultaneous respiratory failure and circulatory collapse. For this reason it is extremely dangerous to administer carbon dioxide to a patient that has been apnoeic for any length of time.

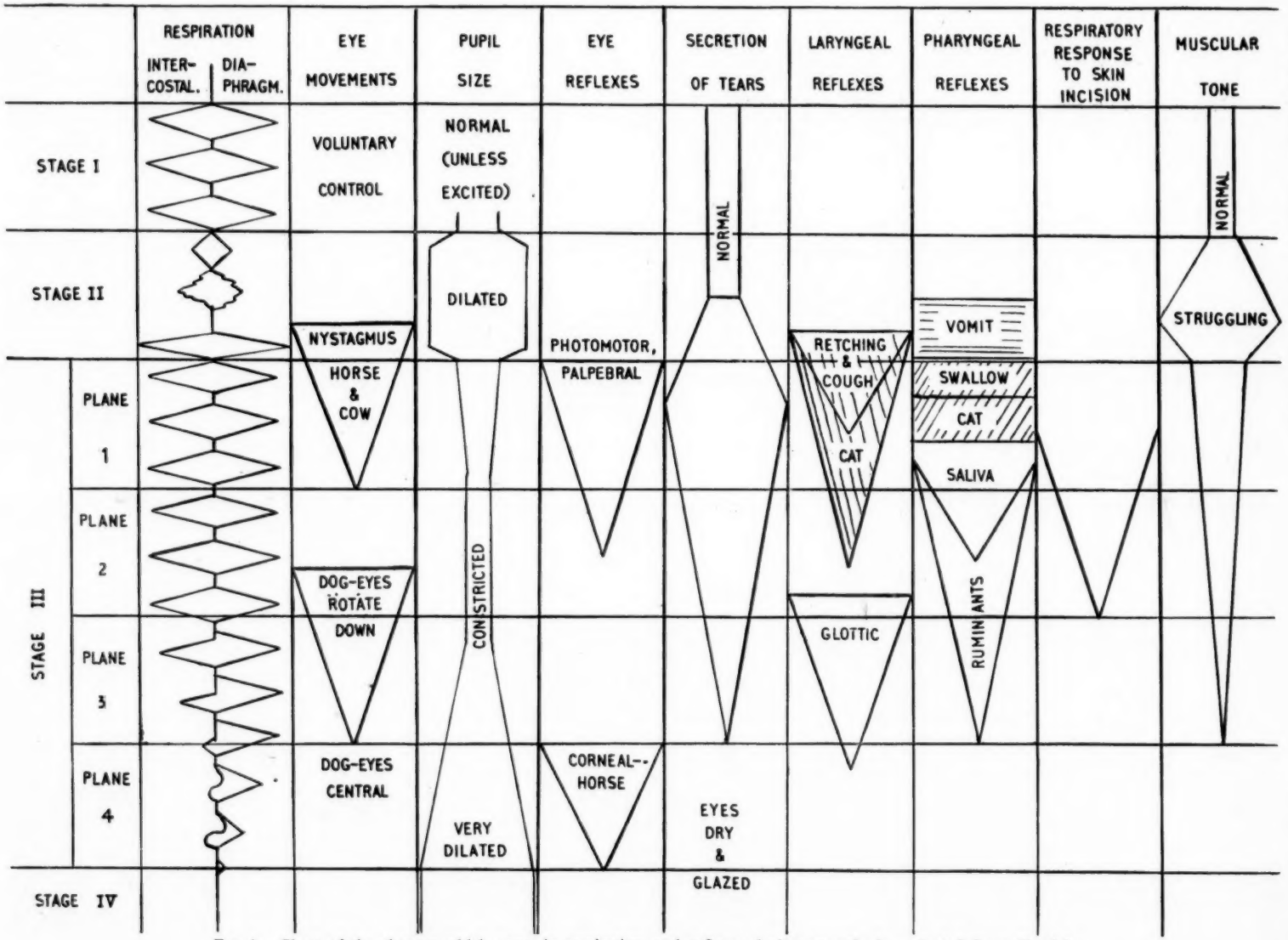

FIG. 2.—Chart of the changes which occur in respiration and reflexes during anaesthesia—adapted from Guedel.

Increased CO_2 level in the inhaled mixture is employed in human anaesthesia in induction with volatile agents, when the stimulation of respiration assists the rapid transition through Stage II. This procedure may be of some value in the cat.

Carbon dioxide levels in the inhaled mixture may rise to dangerous levels in closed-circuit anaesthesia if the soda lime absorber is exhausted and this should be checked before and, if necessary, during administration.

Lowering of the carbon dioxide tension in the blood will produce apnoea by removing the stimulus to the respiratory centre. This fact must be borne in mind when assisting the respiration manually.

Main Action of Anaesthetic Agents

In order that complete control of the anaesthesia of the patient can be accomplished a knowledge of the main action of the different drugs used in inhalation anaesthesia is essential. These will be discussed briefly.

1. *Ether*

Ether vapour is irritant and high concentrations during induction may cause coughing and laryngeal spasm. The laryngeal irritability is increased following barbiturate administration and laryngeal stridor may cause death by asphyxia if pentobarbitone anaesthesia is supplemented by open ether. This effect is especially noticeable in the cat, in which species the laryngeal reflex is very persistent.

Salivation and bronchial secretions are increased. The irritation also causes increased respiratory ventilation and this effect persists to Plane 2 of the third stage. This action, however, lasts only for a period of approximately fifteen minutes. Ether is relatively non-toxic with a wide margin of safety and can be used in closed or open circuit. It is highly inflammable and explosive in mixtures with air or oxygen.

2. *Divinyl Ether*

This agent has similar properties to ether but is more potent, and if used for longer than 30 minutes may cause some liver damage.

3. *Halothane (Fluothane)*

The vapour of Halothane is a highly potent anaesthetic agent and is non-inflammable. It is meantime expensive and only economic if used in closed circuit. It is rapidly eliminated with a consequently short recovery time.

Halothane is a potent respiratory depressant. One of the more important properties of Halothane is its effect on the vasomotor centre, which is markedly depressed, causing a degree of hypotension. This fact is of considerable importance in that the lowered blood pressure may help to prevent shock. Halothane should therefore not be used in conjunction with other hypotensive drugs (*e.g.* chlorpromazine).

No toxic action has as yet been ascribed to Halothane.

4. *Cyclopropane*

Cyclopropane is a gaseous agent which is expensive and uneconomic except in closed-circuit anaesthesia. It forms explosive mixtures with air and oxygen.

It is a powerful respiratory depressant and Planes 3 and 4 of the third stage are short in cyclopropane anaesthesia, so that when the animal is in Plane 2, only a small increase in concentration is required to produce apnoea. This, however, is not a disadvantage since respiratory failure occurs before there is serious depression of the other vital centres, and the animal is not in danger unless apnoea is allowed to persist.

It is important to remember the increase in the respiratory centre threshold when manually assisting respiration by compression of the rebreathing bag. Apnoea can be produced easily in cyclopropane anaesthesia, even in Plane 1, by washing out CO_2 and thus removing the stimulus to the respiratory centre. Cyclopropane increases cardiac irritability and arrhythmias and fibrillation may develop, especially if adrenaline is administered.

The administration of morphine as a sedative has been shown to aggravate this effect on the heart.

5. *Chloroform*

Complete elimination of chloroform may take several days. Chloroform is toxic to the liver, especially if the glycogen content of that organ is low, and if some degree of anoxia is present. Syncope during chloroform induction may take place, associated with breath holding in Stage II followed by excessive inhalation of concentrated chloroform vapour.

6. *Trichlorethylene (Trilene)*

Trilene also decomposes with soda lime and cannot be used in a closed circuit. Volatility of trilene is low so that induction is slow. Trilene is less potent and less toxic than chloroform and has marked analgesic properties but otherwise the two drugs are similar.

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An Enteric Disease of Pigs Resembling Transmissible Gastro-enteritis

BY

P. S. BLIGH

Stowmarket, Suffolk

Introduction

THE following cases are recorded because it was felt that the clinician can aid the investigation of disease by recording the syndromes observed, and because of the similarity to transmissible gastro-enteritis as recorded in America.

Transmissible Gastro-enteritis

As this disease, recorded in America, bears so close a resemblance to the one described in these cases, it was thought worthwhile briefly to review it.

Haelterman and Hutchings (1956) state that transmissible gastro-enteritis is the only primarily diarrhoeal disease of new-born pigs in which viral aetiology has been established. It is probably enzootic in all of the major pig-rearing areas of the U.S.A. It is characterised by a very short incubation period, and by vomiting, diarrhoea, rapid dehydration, and a very high mortality. In older pigs, infection may be inapparent, or it may produce a watery diarrhoea, occasional vomiting, and rarely, prostration. Spontaneous recovery within a few days is usual in animals over 3 weeks old. Lesions are mild to severe inflammation of the gastro-intestinal tract and congestive and degenerative changes in the kidneys. The intestinal tract usually has the highest concentration of virus. It is not affected *in vitro* by penicillin or streptomycin. Some degree of immunity may be transmitted from previously infected sows to their young through the colostrum.

Bay *et al.* (1951) make the following observations on outbreaks studied by them. Young pigs died in 2 to 7 days, older pigs usually recovered in 7 to 10 days. Outbreaks did not usually spread to neighbouring farms. Symptoms in the sow were disinclination to feed, vomiting, and then scouring, loss of weight, dehydration, and if this continued, the sow ceased to lactate. Pigs under 1 week normally died rapidly. They usually developed a diarrhoea, vomited, became dehydrated, emaciated and comatose and died. The faeces of infected pigs had a marked odour and were very fluid. About 90 to 100 per cent. of the pigs affected under 1 week old died. The death rate usually decreased until at 3 weeks of age 40 to 60 per cent. recovered. Occasionally the disease has been observed to cause a few deaths in fatteners and breeding stock.

From their studies, Lee *et al.* (1954), give the following details. The incubation period is very short, 24 hours in older pigs, and as little as 12 hours in young pigs. There is an initial temperature rise which is very short. This is possibly why temperatures taken by the writer were very variable, frequently below normal. Experimentally, pigs could contract the disease when placed side by side in cages without having faecal contact. Affected pigs had a retarded growth rate after recovery. It was found that pigs passed faeces containing virus 21 to 42 days after clinical recovery.

Goodwin *et al.* (1958) record a disease in East Anglia in November 1957, which appears similar to the one to be described. They have also observed its similarity to transmissible gastro-enteritis.

Clinical Description

From June to December 1957, 12 outbreaks of a disease closely resembling transmissible gastro-enteritis have been encountered in this area. Four of the most typical cases have been recorded, and have been selected from breeding herds, as in these diagnosis is easier.

Case 1: Date of Outbreak June 13th, 1957

This occurred in a self-contained herd of 15 sows. At the time of the outbreak 6 sows with litters, 1 pregnant sow and 2 early-weaned litters were housed in a farrowing shed.

The first litter (A) which was 5 weeks old, commenced scouring on Sunday, June 9th. By the following day, 4 more litters (B, C, D and E) were scouring. The sixth litter (F) was unaffected. The "in-pig" sow (G) farrowed on the 15th and her litter was affected at 48 hours of age. Neither of the weaned litters was affected. The first death occurred 72 hours after the onset of the outbreak, and deaths continued for 5 days as follows: 7, 8, 4, 3, 3, per day (see Table I).

TABLE I

Litter	Date of onset	Age of litter	Number in litter	Number of deaths
A	9.6.57	5 weeks	9	0
B	10.6.57	,,	12	0
C	,,	3 ,,	9	6
D	,,	10 days	12	12
E	,,	,,	7	7
F	unaffected	5 weeks	–	–
G	17.6.57	48 hours	11	9
			60	34

The scouring persisted up to 1 week in pigs which recovered. No scouring was observed in the 6 suckling sows, but 5 showed inappetence for periods up to 5 days. Eight dry sows and a boar which were kept in an orchard were unaffected.

The outbreak was first seen on June 13th, by which time there had been 10 deaths. Small pigs were given sulphadimidine subcutaneously. The faeces were very fluid, mucus speckled and having an odour resembling that of a summer mastitis. The scour continued until death or recovery.

By the next day there was no apparent improvement, and a further 6 deaths had occurred. Sulphadimidine was given orally and an *Escherichia coli* polyvalent anti-serum was administered subcutaneously. When the herd was next seen on the 17th there had been a further 5 deaths and no improvement. The litter of sow G (see Table I) had now commenced to scour. Temperatures of the sow and litter were taken and were as follows: sow 104.5; 10 piglets 99.8—102.2 and 1 piglet 104.2° F.

Laboratory examination failed to reveal any organisms of significance. Histological examination of the brain of 2 piglets and Gel precipitin tests on the pancreas of 4 others gave no evidence of swine fever infection.

Case 2. Date of Outbreak July 16th, 1957

This herd comprised 18 breeding sows and about 60 fattening pigs; 5 of the sows were suckling. The litters varied in age from 5 days to 4 weeks. All the sows with litters were under one roof. The remainder were at grass.

On the first visit 3 sows were ill and scouring slightly, but less violently than in Case 4. The temperature of one sow was 104.0° F., the other 2 had normal or sub-normal temperatures, the lowest being 101.5° F. All the piglets were scouring badly. Eight of the youngest piglets had died. The losses followed a similar pattern to those in Case 1 but were heavier, possibly because the piglets were younger, 32 dying out of a total of 54 pigs (see Table II). No temperature rise was observed in the piglets.

TABLE II

Age of litter	Number in litter	Number of deaths
5 days	10	10
10 „	12	10
2 weeks	12	8
4 „	11	3
4 „	9	1
	54	32

The fattening pigs were in one fattening house and were divided into 5 different lots, 4 of which were scouring. In the pens containing scouring pigs, *all* the animals appeared to be affected.

Treatment of the small pigs consisted of sulphadimidine and streptomycin given orally, but this had no effect. The fattening pigs were dosed with sulphadimidine by mouth. About 75 per cent. showed a marked improvement by the next day. The remainder took about a week to improve with the exception of 2 animals which died. The sows at grass remained unaffected as in Case 1.

Case 3. Date of Outbreak December 3rd, 1957

This was a small herd consisting of 3 "in-pig" sows (one of which farrowed the day following the first visit), 12 10-week-old pigs, 10 12-week-old pigs, and 8 pigs ready for the bacon factory. There had been no recent purchases.

When first seen all the pigs were scouring with the exception of 2 of the sows. The sow affected was scouring badly and had not eaten for 2 days. Her temperature was 100° F. One of the 10-week-old pigs had died, 1 was moribund and 2 were ill. Scouring commenced the previous day and affected pigs were inappetent. The temperatures varied from 100° to 104° F. *Post-mortem* examination revealed a haemorrhagic gastro-enteritis.

The affected sow was given sulphadimidine subcutaneously. All the others were dosed by mouth, with the exception of the bacon weight pigs, which were sent for slaughter the following day.

The next day, the scouring had reduced considerably. The sow still would not eat, and a further sow was affected and was treated accordingly. All pigs with dirty tails and those "tucked up" were dosed again. The heavily "in-pig" sow was apparently still healthy, and farrowed 10 healthy young pigs. One of the bacon pigs died before arrival at the bacon factory, and 1 of the sick 10-week-old pigs died, but neither revealed any points of interest at *post-mortem*.

On the third day the remaining sow and her litter commenced to scour, and another of the sick pigs had died. The 24-hour old piglets were lethargic and 1 had died. The dam appeared to have adequate milk. The sow was injected with sulphadimidine and the litter given sulphadimidine by mouth. Only 3 of these survived until the following day—they were scouring badly and soon died.

There were no further deaths. The first sow to be ill took 5 days to return to her food. This was achieved by putting her to grass for 2 days. She then fed normally. The other 2 sows were only ill for one or two days.

Case 4. Date of Outbreak December 12th, 1957

This outbreak occurred in a herd of about 200 animals, comprising about 40 sows, 30 gilts and 15 boars awaiting sale, and approximately 100 pigs whose ages varied from 3 to 12 weeks.

Four to 5 days before my visit, 1 sow with litter had shown inappetence. Two days later she and another sow commenced to scour badly. These 2 sows were in the same farrowing shed, which contained 3 other sows and litters. The following day, a further sow went off her food, and according to the pigman, this was when he noticed "undue" scouring among the sucklers, which varied in age from 3 to 7 weeks.

Examination revealed that the younger the litter, the more severe the disease. Four of the 3-week-old pigs had died, and 1 was moribund. They were dehydrated and weak on their legs, and faeces had the consistency of water, and were brownish-white in colour. Not much importance was attached to colour, as possibly this was related to the amount

of creep feed and milk consumed. Pigs which were almost wholly fed on solid food had mud coloured diarrhoea with an unpleasant odour. Every pig in these 5 litters was scouring and appeared lethargic.

There were 6 other sows with litters, 4 in one building and 2 in another. In the large house, 2 of the sows were slightly affected, and the litters had a slight diarrhoea, but they were still feeding and appeared bright and alert.

The adult stock in the bullock yards was examined, and 75 per cent. of these were scouring and about 50 per cent. off their food. The pigs in the field were apparently normal.

All the affected pigs were dosed with sulphadimidine by mouth, and this had a very beneficial effect, as the only further death was the badly affected 3-week-old pig. Most of the pigs had stopped scouring by the following day. The non-affected litters were not dosed and remained unaffected. No adult stock was dosed.

Adult stock which did not return to its food within 3 days was put out to grass, which was often eaten ravenously. In other cases, sugar beet tops were fed, and this treatment restored their appetites. Sows which were brought in from grass to farrow, were brought in about 14 days to 3 weeks before due, as normally if they were to be affected, it occurred 3 to 4 days after arrival, and recovery usually occurred about 2 to 5 days later. When recovery had occurred, litters did not seem to be affected. No sows farrowed within 14 days of the outbreak. After about 10 days, the sows in the field were affected, very mildly. They were not inappetent.

One point of interest in this outbreak was that 2 of the sows vomited.

While this outbreak was in process, the next door farm, which had 45 3-month-old pigs in 3 pens of 15, had a mild outbreak of scouring, but this was not considered typical of the condition as only 50 per cent. of the pigs in each pen scoured. A month afterwards the farm on the other side, which had 3 sows with litters varying in ages from 6 to 8 weeks, reported that 2 of the sows had gone off their food. Three days later they commenced to scour, as did the third sow. On the fourth day, the litters scoured, but started to recover within 24 hours. These piglets which had received no treatment were seen 6 weeks later, with a history of intermittent diarrhoea and unthriftiness. Sulphadimidine given orally produced some noticeable improvement.

These latter cases would not normally be diagnosed as the condition being described because of their mild nature, but owing to their location and similarity, it is possible that they may have been but had a variation in severity. This, particularly in non-breeding herds, would make diagnosis impossible on clinical grounds.

Observations on the Outbreaks

Of the 12 outbreaks examined several cases were suspected in flying herds, and diagnosis was based on the following:—

1. When a pen was affected, every pig in that pen scoured within about 12 hours.
2. The younger pigs were more severely affected.
3. The disease normally reached its climax in one fattening house in about 5 to 7 days, when recovery was usually fairly evident.
4. Losses were small in weaned pigs.

It is possible that these conditions could be varied slightly, but if such a margin is allowed several other causes of scouring would be included.

Symptoms

These were as described in the case histories. Thirst in some instances was very marked, in fact in one case the pigs attempted to catch raindrops. This was mainly due to the feeding which was dry, with restricted water intake. The scour dehydrated the pigs who had no opportunity of replacing their fluid loss. It was felt that in this particular outbreak some deaths occurred through dehydration, because when water was given *ad libitum* some improvement was noted.

Vomiting was not a common symptom, in fact only about 3 to 4 instances were recorded, and none was personally observed.

The general symptoms were inappetence, evil smelling diarrhoea, dehydration and in some advanced cases, ataxia.

The writer had the opportunity of comparing a case of swine fever with Case 3, as both were being dealt with concurrently. The main difference appeared to be that in Case 3 the spread was far more rapid, and commenced to resolve in 1 to 2 days. There was only a short temperature rise which usually passed unnoticed. In a large outbreak the condition can last for several weeks, because of the occurrence of fresh cases, but at the same time there is always a large number of recovered and recovering cases. The growth rate of recovered pigs varied. In some outbreaks there was stunting of the type which often follows "paratyphoid," but this was not so evident when pigs had been treated with sulphadimidine.

Treatment

In small pigs, under 2 weeks of age, treatment appeared to be of little value. Older pigs seemed to benefit from the use of sulphadimidine given orally. The sodium salt was injected into the mouth with a syringe to make certain the dose was taken. This treatment was more effective when used the day the diarrhoea commenced. The variation in response may have been due to the fact that the sulphadimidine affected only secondary bacterial invaders prevented the normal bowel flora from becoming pathogenic due to decreased resistance of the intestine as it seems likely that the primary cause is a virus. Inappetence in adult stock was often overcome by putting them out to grass. Sows which had not eaten for 4 or 5 days would often eat greedily when this was done. Some degree of control was achieved by bringing in farrowing sows about 14 days before they were due. The sows then developed symptoms and recovered and their subsequent litters were frequently immune.

(*Concluded at foot of col. 1 overleaf*)

An Experimental Outbreak of Husk Among Previously Parasitised Cattle

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AND

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SUMMARY.—A group of young cattle were exposed to lungworm infection on pasture and subsequently withheld from infection. In the following year they were again exposed to infection on pasture together with a group of cattle of the same age which had been housed since birth. The resulting symptoms of husk were of equal severity in the two groups but lungworm larvae appeared only in the faeces of the cattle of the group which was exposed to infection for the first time.

Introduction

IN an earlier communication (Michel & Shand, 1955) circumstantial evidence was discussed derived from the study, after the event, of outbreaks of husk in adult cattle. In most cases it appeared that the outbreak was not the consequence of an introduction of infection into a worm-free herd. It was suggested that the parasite could persist on a farm for many years at a subclinical level and an unusual combination of circumstances was necessary to produce an outbreak, the essential feature being the sudden exposure to infection of animals which had experienced a negligible intake of infection for some time.

A striking feature of many outbreaks among adults was that larvae were often absent from the faeces and that in animals which died, the worms recovered from the lungs were immature and few in number. Subsequent laboratory experimentation had shown that when infective lungworm larvae were administered to animals which had recovered from a previous infection, symptoms could occur (Michel, 1954). No larvae appeared in the faeces of such reinfected animals, however; a circumstance apparently due in part to the inhibition of development of the worms initially established (Michel, 1955). There is also some evidence to suggest that some of the worms initially established are rapidly lost from the lungs.

These findings supported the conclusion that most husk outbreaks in adults occurred in animals which had had previous experience of infection. Michel and MacKenzie (1956) were able to confirm that sudden exposure to infection is necessary, the infections in their experimental cattle being entirely referable to larvae picked up over a very short space of time. This phenomenon was due to the rapid development by the animals of the ability to prevent the establishment of newly acquired worms, and Michel and Parfitt (1956) showed that the fate of a calf grazing on an infected pasture depended only on the level of the herbage infestation during a short initial period, the animal being unaffected by subsequent increases in herbage infestation, no matter how great. The circumstantial evidence of Michel and Shand (1955), however, together with the results of experiments on housed calves, suggested that the ability to resist the establishment of worms tends to wane with the passage of time and it seemed desirable to determine whether an outbreak of husk could indeed be produced by the re-exposure to infection of resistant cattle which had been withheld from infection for some time. The opportunity arose to incorporate such a situation in the design of a series of experiments at the National Agricultural Advisory Service Experimental Husbandry Farm at Boxworth, near Cambridge.

Methods and Results

Thirty-six Friesian calves which had been segregated from their dams shortly after birth and reared in the buildings were assigned at random to 3

An Enteric Disease of Pigs—*Concluded.*

Post-mortem Findings

These were variable, a haemorrhagic gastro-enteritis being the most constant finding.

Conclusion

A condition which causes considerable economic loss among suckling pigs, and to a lesser extent in fattening herds, has been described. The writer feels that it may be widespread but has not been more frequently reported because of the difficulty in reaching a specific diagnosis and of differentiation from other enteric conditions of pigs.

Apologies are given for the lack of concrete evidence in the form of laboratory reports, but the few which were obtained failed to throw any light on the cause.

Acknowledgments.—To the Investigation Department, Cambridge, for their advice on the earlier cases investigated and for carrying out laboratory examinations.

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groups, A, B, and C. On May 28th, 1955, the calves of group A, which were now approximately 6 months old, were put onto 3 acres of a fifth-year timothy-meadow fescue-white clover ley on which 4 experimentally infected Ayrshire calves were also grazing. These Ayrshire calves had received a dose of 3,000 larvae of *Dictyocaulus viviparus* on May 5th and now served as a source of infection. The herbage infestation rose to a peak of 0.6 larvae per lb. of herbage and on July 8th the experimental cattle were removed from the infected pasture. They were now withheld from infection by a system of close folding with the electric fence, over pastures which had carried no cattle for at least 6 months, and moving to a fresh strip daily. That this procedure was successful may be seen from the fact that, in their strip grazing, the animals of group A were mixed with a group of clean cattle, group C, whose mean faecal larval counts at no time during the season exceeded 0.1 larvae per gramme. That this figure should be so low was gratifying in view of the fact that only very limited precautions could be taken to avoid the carriage of infection onto the clean pasture on the wheels of vehicles or the boots of stockmen. Moreover, the cattle were moved at regular intervals and by the same route to the buildings where they could be weighed and faeces samples taken.

Moderate symptoms of husk appeared in group A by the end of the first week in July. Coughing was obvious but not painful and though respiratory rates were above normal only one animal showed signs of dyspnoea. Nonetheless the cattle suffered a loss in weight of some 20 lb. per beast as compared with the control group C which loss, however, they gradually made up in subsequent months. Symptoms were subsiding by the end of August.

On September 9th, there being no more clean pasture available, the cattle were housed in a covered yard where again the chances of accidental infection were negligible, especially as none of the cattle were by this time passing larvae in their faeces.

Re-exposure in the following year had to be undertaken under conditions of strip grazing. Four Ayrshire calves of which two had received 3,000 larvae and two 4,000 larvae on April 19th were put on the first strip on May 21st, 1956, and moved to a fresh strip daily. The experimental cattle were brought out of the yards and began to proceed from strip to strip on May 28th so that they continued to occupy ground on which the Ayrshire calves had been 7 days previously. In this way, by means of frequent herbage sampling, it was possible in some measure to control the severity of the infection acquired by the experimental cattle. The infection process was allowed to continue for 12 days, the mean herbage infestations to which the cattle were exposed during this period being 0.69 larvae per lb. of herbage. Thereafter the cattle were close-folded over clean ground as in the previous year.

The cattle which were simultaneously exposed to infection in this manner now consisted of group A, previously infected in 1955, group B of the same age but housed since birth, and a new group D consisting of ten Friesian calves approximately 6 months old which had also been housed since birth.

Coughing became obvious in groups A and B during the first week of June. The symptoms were of much the same severity and duration as in the previous year but no difference could be detected between the two groups. Any animal which showed signs of respiratory distress or whose declining bodily condition gave cause for alarm was put into the buildings, where it usually remained for a period of a fortnight. When the records were subsequently examined it transpired that of the nine animals housed in this manner 5 were from group A and 4 from group B.

Meanwhile the calves of group D showed very mild symptoms and even when they were moved hardly any coughing could be heard. Faecal larval counts of the experimental cattle are shown in Table I. It will be seen that in 1956, larvae appeared in the faeces of groups B and D but were absent from the faeces of group A. (Table I appears overleaf.)

Discussion

It has been shown that if cattle have continuous access to infection with lungworms, then after a relatively short initial period even the greatest increase in herbage infestation does not give rise to symptoms (Michel & Parfitt, 1956). In contrast the results reported here show that resistant cattle which have been withheld from reinfection will in time again become susceptible and that clinical symptoms can result of equal severity to those observed in cattle which have had no previous experience of infection. In the observations recorded the infections in the previously parasitised cattle did not become patent, a circumstance which has also been observed in many outbreaks of husk in adult cattle (Michel & Shand, 1955) and which may be attributed to the inhibition of development of the worms in the resistant animal and to their failure to persist. The various manifestations of resistance to nematode infections should be regarded as separate and distinct one from another in their effects, their causation, and their persistence. In the case of lungworm infections in cattle, it appears that though the ability to resist the establishment of worms declines with the passage of time, the ability to inhibit the development of the worms persists as does the ability to eliminate them from the lungs. Thus, in the spring of 1956, the resistant cattle of group A, though they were susceptible to the initial establishment of worms, were still able to prevent the new infection from becoming patent.

A number of indications regarding the relationship between animal management and outbreaks of husk in adult cattle emerge from this work. In the past most thinking on the subject has been based on the assumption that outbreaks are the outcome of a protracted build-up of infection as between the pasture and the cattle. In consequence the measures usually advocated are aimed at preventing animals from reinfecting themselves. It is now clear that this aim is incorrect and that to deny cattle access to infection introduces a real hazard in that it renders

TABLE I

FAECAL LARVAL COUNTS OF THE EXPERIMENTAL CATTLE EXPRESSED AS LARVAE PER 10 GM. FAECES

	1955							1956								
	May 6th	June 10th	July 7th	July 21st	Aug. 11th	Aug. 26th	Sept. 13th	May 24th	June 21st	July 4th	July 18th	Aug. 1st	Aug. 15th	Aug. 29th	Oct. 10th	Nov. 21st
Group A																
629	—	—	1	417	1	—	—	—	—	—	—	—	—	—	—	NS
633	—	—	2	250	5	—	—	—	—	—	—	—	—	—	NS	NS
661	—	—	2	120	1	—	—	—	—	1	—	—	—	—	—	NS
679	—	—	16	1,730	8	—	—	—	—	—	—	—	—	—	NS	NS
618	—	—	3	474	58	—	—	—	—	—	—	—	—	—	—	—
639	—	—	41	350	9	—	—	—	1	—	—	—	—	—	NS	—
650	—	—	2	520	8	—	—	—	—	—	—	—	—	—	—	1
651	—	—	1	480	NS	—	—	—	—	—	—	—	—	—	—	—
666	—	—	—	600	16	—	—	—	—	—	—	—	—	—	—	—
682	—	—	5	505	—	—	—	—	—	—	—	—	—	—	—	—
690	—	—	31	875	361	—	—	—	—	—	—	—	—	—	—	—
698	—	—	3	456	4	—	NS	—	—	—	—	—	—	—	1	—
Group B																
642	—	—	—	NS	—	—	—	—	—	80	2	—	—	—	—	NS
648	—	—	—	NS	—	—	—	—	—	NS	—	—	—	—	—	NS
659	—	—	—	—	—	—	—	3	—	5	—	—	—	—	—	NS
681	—	—	—	—	—	—	—	—	—	1,970	—	—	—	—	—	NS
636	—	—	—	—	—	—	—	—	—	402	1	—	—	—	—	—
637	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—
652	—	—	—	—	2	—	—	—	3	6	1	—	—	—	—	—
663	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
665	—	—	—	—	—	—	—	—	—	312	65	4	—	1	—	—
667	—	—	—	—	—	—	—	—	—	233	13	—	—	—	NS	—
695	—	—	—	—	—	—	—	—	—	1,095	139	—	—	—	—	—
711	—	—	—	—	—	—	—	—	—	75	—	—	—	—	—	—
Group D																
728								—	—	10	—	—	NS	—	—	NS
737								—	—	92	5	—	—	—	1	—
748								—	1	8	—	—	—	—	—	—
783								—	—	415	16	—	—	—	—	—
791								—	—	142	41	—	—	—	—	—
735								—	—	600	8	—	—	—	—	—
760								—	—	565	60	1	—	—	NS	—
766								—	NS	36	—	—	—	—	NS	—
775								—	—	268	72	15	—	—	—	—
776								—	—	167	32	NS	—	—	7	—

them susceptible to the consequences of reinfection.

Dangerous herbage infestations need not be the result of heavy and prolonged contamination of the pasture with infected faeces, indeed experiences at Weybridge show that a lightly infected animal can, in favourable conditions, produce lethal herbage infestations on large areas of pasture in a matter of a few days. A policy of withholding cattle from infection should therefore be attempted only if it can be consistently carried through, a course which would imply a move, at least every third day, to pasture which had not been grazed by cattle for 3 summer months or 5 winter months. Such a procedure could hardly be contemplated on the average farm.

It is evident from these results and those of Michel (1957) that control measures must have for their aim that first exposure should be to a low herbage infestation and contact with infection should thereafter be continuous.

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COW LIVES FOR SIX WEEKS WITHOUT FOOD

A Pretoria veterinary surgeon describes as "a miracle" the survival of a cow in a hole for six weeks with not even enough food for one day.

The cow vanished not long after being bought. An extensive search was made, but the cow could not be found.

Six weeks later, within 100 yards of the farm-house, she was seen in a hole about three feet deep. The grass around the edges of the hole had been ate down to the ground and as she turned the cow had made a groove in the sand walls of the hole.

Caesarean Operation on a Mare

BY

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A CAESAREAN operation was recently performed on a grey Thoroughbred mare—"Smart Girl" by "To You II" out of "Fashionable." The mare, owned by Mr. M. Englebrecht of the farm "Onzerust" in the Barkly East district of South Africa, is the dam of three winners.

Reason for Performing the Operation

The mare was suffering from dystokia, caused by the lumbo-pubic position of the foetus, with the fore legs flexed and the head directed backwards. The foal was dead (*rigor mortis* having set in), and both the vulva and the vagina were swollen. It was impossible to correct the position of the foal in spite of epidural anaesthesia.

Anaesthesia

Two and a half oz. of chloral hydrate were dissolved in ½ gal. of water and introduced through the stomach tube. The operative area on the right flank was infiltrated with 40 ml. of 3 per cent. procaine hydrochloride.

Control of the Animal

The mare was left in the lateral position in which she was lying—being unable to rise. The fore legs were stretched forward and secured with a rope, and the hind legs were stretched backwards, and held by two natives, while a third native held the head.

Technique

After preparing the operative field and the surrounding area by shaving and disinfecting with Dettol, an incision about 15 inches long was made through the skin, abdominal muscles, and the peritoneum. The bulging-out intestines were held down effectively by the farmer who was assisting.

The hind legs of the foal were located through the wall of the uterus, and by holding these, part of the uterus was pulled out. An incision about 12 inches long was then made through the wall of the uterus, and the foetal membranes were torn open, and the foal removed. The uterus tore along the incision for a further 4 inches during the process of removing the foal.

Then, after removing the loose parts of the placenta, 2 oxytetracycline pessaries and approximately ½ oz. of sulphanilamide powder were introduced into the uterus.

The incision in the uterus was closed with continuous Lembert sutures and 2 rows of interrupted sutures using No. 3 chromic catgut (20 days). The sutured incision was then dusted with sulphanilamide powder. The abdominal muscles and the peritoneum were stitched with a row of interrupted sutures, using No. 3 catgut doubled. The subcutaneous muscle was stitched separately also using interrupted sutures. The skin was then closed with 5 mattress sutures and the edges finally closed with interrupted sutures. A piece of gauze was then stuck on to the area as a protection against flies.

Thirty international units of oxytocin were injected intramuscularly and 3,000,000 units of procaine penicillin G in aqueous suspension were also injected.

The mare rose immediately upon being released.

Subsequent History

Three days after the operation the author carried out an examination on the mare which was grazing at the time. Her temperature was normal, but a swelling in front of the udder was apparent, and this the author presumed to be due to histamine compounds from the retained part of the afterbirth. Intramuscular injections of 3,000,000 units of penicillin and 3 ml. of stilboestrol were administered.

The owner of the mare was instructed to inject 5 ml. of 5 per cent. promethazine-hydrochloride solution for the next 2 days, and thereafter 2 g. of dihydrostreptomycin and 3,000,000 units of penicillin alternatively for the following 4 days. Altogether the mare received 12,000,000 units of procaine penicillin G and 8 grammes of dihydrostreptomycin.

The author again visited the stud 7 days after the operation, and removed the mattress sutures. The temperature was normal and the mare was grazing contentedly. The owner reported that the remaining part of the placenta had been passed 2 days previously.

Eight days after the operation the mare was on heat.

On the tenth day after the operation the last stitches were removed.

Remarks

The operation described above was performed in the open veld under the blazing South African sun, while the wind occasionally caused dust, hairs, and dry grass to settle on the wound. The Europeans and natives who assisted were quite untrained and inexperienced, so that it was difficult to get them to observe the rules of asepsis. The mare was 14 years old and had spent the night in labour. The author was unprepared for the operation, and consequently had no choice of anaesthetics—his surgery being 20 miles distant. There was need for haste in performing the operation as it was impossible to provide any kind of shade, and the blazing sun caused the blood to dry rapidly.

It has generally been accepted that laparotomy on a horse is dangerous because of the risk of infection ani peritonitis, and because of the difficulty in handling the considerable amount of bulging intestines.

Thus, taking the unfavourable and difficult conditions described above into account, it would appear that there is no appreciably greater risk attached to laparotomy in a horse than there is in other animals, such as sheep, cows, etc. The author frequently performs caesarean operations on sheep and cows, and he considers that all in all there is not much more work attached to carrying out this operation on horses.

Addendum

The mare was served on December 25th and 28th, 1957, about 7 weeks after the operation, and 6 weeks later a blood sample was sent to the Veterinary Research Institute at Onderstepoort. The result was positive and proved that the mare was again in foal.

Current Literature

BOOK REVIEWS

Veterinary Pathology. By H. A. SMITH & T. C. JONES. Pp. 959. Figs. 263. Henry Kimpton, London, 1957. £6 10s.

An entirely new book on veterinary pathology must inevitably be the subject of critical interest to many readers concerned with the various medical sciences, and, although as the authors state in the preface, this book is intended primarily for the veterinary reader, they also hope it will be of value to the comparative pathologist. In the opinion of this reviewer the authors' claim to the attention of a wider audience is more than justified.

The first 8 chapters are concerned with those broad changes covered by the term General Pathology, including cellular degenerations, deposition of minerals and pigments, disturbances of growth and circulation, inflammation and body reactions, neoplasia, and the causes and nature of disease. In this part of the book the chapter on neoplasia is outstanding and the illustrations particularly helpful and to the point. The chapter is concluded by a discussion on the incidence of neoplasms which includes a most useful table compiled from the authors' own records, and those of Cotchin in this country and Jackson in South Africa. It is based on a total of 7,052 tumours classified by location and kind of neoplasm and the species in which they were present.

On the other hand one must admit to a certain feeling of disappointment after reading the contribution on inflammation. The histological changes are adequately described but the underlying mechanisms receive little detailed exposition, and much of the biochemical background is ignored. There is a tendency to teleological description of inflammatory processes as in the account of the margination of leucocytes in blood-vessels which reads: "They appear to be searching and examining every inlet and bayou of the swollen and roughened capillary lining as if, like houseflies on a screen-door, they were looking for a way to get through. But let us say merely that they have developed some quality of adhesiveness toward the vessel wall, for we do not attribute intelligence to these cells. Or should we say that they are drawn toward the outside by some irresistible force, as a lover is drawn to his lady fair?" Descriptive, perhaps, but hardly explanatory.

The next 5 chapters are devoted to the pathological effects of viruses, rickettsiae, bacteria, fungi, and spirochaetes as well as protozoa, parasitic helminths, and arthropods. The chapter on diseases caused by viruses and rickettsiae is particularly valuable, and deals with a gap that has been neglected or avoided in other text-books. It is this chapter which is accompanied by a magnificent coloured plate illustrating cellular inclusions of viral infections. The chapters on bacterial infections and fungi reflect the North American interest in problems which as yet do not trouble us, and British readers may feel that conditions such as Johne's disease receive rather sketchy treatment and braxy and lamb dysentery will not even be found under clostridial infections or diseases of the digestive system. Chapters on the pathological effects of ionising radiations and diseases due to extraneous poisons are, unfortunately, particularly welcome for the problems arising from these risks are becoming more widespread.

The chapters on the diseases of the various systems are comprehensive, but the veterinary student may find the systems of classification, especially on disorders of liver and kidney, need some adaptation to that which is taught in British schools. Each chapter is accompanied by a selective but helpful key bibliography.

These criticisms are trivial for the authors have produced a new text-book of outstanding value. The text is clear, readable, and as comprehensive as possible. A striking feature of the work is the first-class illustrations which without exception illuminate the text at every stage, and their presence in themselves make this book a landmark in veterinary literature. Most pathologists will want access to this book, and it is to be hoped that its price will not deter too many students, for it has much to offer to its readers.

The Clinical Application of Antibiotics. Vol. III.—Chloramphenicol and the Tetracyclines. By M. E. FLOREY. Pp. ix + 393. Oxford University Press, 1957. 84s.

This is a continuation of the earlier work, "The Clinical Application of Antibiotics—Penicillin," which was published in 1952 and is to be considered as Volume I of the series. The aim is to evaluate critically the data on which the present use of antibiotics is founded. The importance of accurate diagnosis is therefore stressed throughout for on this alone can a true evaluation be made. About 100 pages are given to the general consideration of chloramphenicol and its use in diseases due to specific organisms; the descriptions of its use in diseases in which a variety of organisms may be responsible are brief and limited. The reason given is that reliable laboratory data should be available before chloramphenicol is given and it is essential that no other drug is expected to accomplish as much. Serious consequences have sometimes followed its administration.

The tetracyclines which include aureomycin, terramycin, and achromycin are considered in a similar manner. The treatment of diseases due to specific organisms precedes the account of their uses in diseases associated with varying organisms and classified according to the bodily systems attacked.

A glossary of bacteriological nomenclature, a bibliography of more than 40 pages, and an index complete this volume.

Primarily meant for students of human medicine, this should be valuable to those concerned with animal diseases.. The critical evaluation which is the recurrent note is as applicable to veterinary sur-

(Concluded at foot of adjoining column)

ABSTRACTS

Early Farming Transfer Charm Cures. DAVIDSON, T. (1957). *Agriculture, Lond.* **64.** 458-61.

Until comparatively recent times, diseases of farm stock were still being treated by superstitious practices inherited from the dark ages. The author describes various rites and charms by which, it was believed, diseases could be transferred from sick animals to other animals or to some inanimate object, such as the ground. These practices ranged from the burial alive of the first animal to be affected to the less revolting custom of turning over the first sod trodden in the morning by a lame horse or ox. The article should be seen in the original for a full account of these various practices.

E. A. G.

Successful Method of Destroying Large Numbers of Turkeys Under Field Conditions. BODENWEISER, L. E., & PETERSON, K. J. (1958). *J. Am. vet. med. Ass.* **132.** 238-40.

The authors describe the destruction in Oregon, U.S.A., of a flock of some 2,500 adult turkeys affected with ornithosis (psittacosis). Two long pits were dug with a bulldozer, the birds driven in, covered with polythene sheeting, and gassed with car exhaust fumes. Euthanasia was complete within 8 to 10 minutes. There was no excitement or struggling. As the birds were not handled the risk of infection to the operators was minimal.

Aspergillus Meningo-encephalitis in Turkeys and Ducks. HUBBEN, K. (1958). *Avian Diseases.* **2.** 110-16.

The author describes 2 outbreaks in which 2½- and 3-week-old turkey poults, kept on deep litter, showed a variety of nervous symptoms including torticollis, retropulsion, and ataxia. *Post-mortem* examination revealed typical aspergillotic lesions of the air sacs and lungs, together with brain lesions. A third outbreak occurred among Eider ducklings in a zoological garden.

Preliminary Observations on Haemorrhagic Enteritis of Turkeys. GALE, C., & WYNE, J. W. (1957). *Poult. Sci.* **36.** 1,267-70.

The authors described 2 outbreaks of this condition. Both subsided without treatment. One, which affected 7-week-old turkeys, subsided in 24 days after causing a mortality of 1.1 per cent. The other, in 11-week-old birds, lasted 21 days and caused a mortality of 3.5 per cent. Symptoms were rarely noticed. *Post-mortem* findings were largely confined to the intestines, which contained bright red blood throughout their length. The duodenum was especially inflamed. In a few cases the liver and/or kidney showed petechial haemorrhages. The cause remained obscure.

Studies on the Vitamin E Requirements of Turkeys for Reproduction. JENSEN, L. S., & MCGINNIS, J. (1957). *Poult. Sci.* **36.** 1,344-50.

The fertility of eggs from turkey hens deficient in vitamin E was unimpaired, but their hatchability was generally reduced—in some cases greatly so. Five per cent. of poults from deficient hens showed a cloudiness of the central portion of the lens and were apparently blind. Poults without this defect showed normal weight gains on a practical ration.

E. A. G.

The Incidence of Arthritis in Swine following Vaccination for Swine Erysipelas. NEHER, G. M., SWANSON, C. B., DOYLE, L. P., & SIKES, D. (1958). *Amer. J. Vet. Res.* **19.** 5-14.

Experiments were carried out on 96 pigs, of which 66 were vaccinated. Of these, 53 had received the aluminium hydroxide absorbed type, and 13 the desiccated live culture and serum. The 30 non-vaccinated were used as controls. All the pigs were from 3- to 4-month-old Chester-whites. The pigs were subjected to infections from 6 weeks to 4 months after vaccination. The intravenous method of applying infective material proved a better way of producing infection than by contact or skin scarification. Although the previous vaccination did not prevent the infection, it did stop the high mortality which was found in the controls. Vaccination will not protect the pigs against arthritis: of the 50 protected animals exposed to infection intravenously, 31 (62 per cent.) had arthritis 2 months after being infected. Only one (14 per cent.) of the 7 surviving unvaccinated pigs developed chronic arthritis.

Sensitisation may be important as marked anaphylactic reactions were seen in the vaccinated pigs at the time of injection, in contrast to the negative or only slightly allergic responses in the controls. The production of arthritis in pigs by repeated exposures to *E. rhusiopathiae* seems analogous to the arthritis arising naturally in pigs which have been clinically ill from acute swine erysipelas. In view of the wide distribution of the organism in species other than swine, and in healthy swine, it seems probable that some pigs may develop arthritis under farm conditions through sensitisation by multiple natural exposures.

D. J. A.

Book Reviews—*Concluded.*

geons as to medical practitioners. Results of clinical trials, accounts of experimental work with small animals, as well as *in vitro* tests and reports of individual treatments are collected, sifted, and discussed. There is thus collected into one volume an immense record of study and thought.

INTERNATIONAL SCIENTIFIC FILM ASSOCIATION CONGRESS

We have been advised that forms for films proposed for submission to this Congress, which is to be held in Moscow from September 10th to 20th, 1958, must reach the organising committee by July 15th. A few forms are still available and may be obtained from Colonel G. V. Short, Veterinary Department, Imperial Chemical Industries, Ltd., Fulshaw Hall, Wilmslow, Cheshire. In view of the short time remaining, those wishing to exhibit films are advised to write to Colonel Short immediately.

In Brief

Gram-negative Polysaccharides and Pathogenicity

The presence of polysaccharides in bacterial cells has been known for many years but it needed the precise analytical work of Heidelberger, Goebel and Avery (1923, 1924, 1925) on the specific capsular polysaccharides of pneumococcus to focus serious attention on these highly important materials.

Since this discovery much work has been done to increase our knowledge of these substances, and it is now confirmed beyond doubt that polysaccharides play essential roles not only in bacterial antigenicity, but also in pathogenicity.

Polysaccharides are present in bacterial membranes, capsules and cytoplasm. They occur in gram-positive and gram-negative organisms but representatives from each group show significant chemical and biological differences. Polysaccharides from gram-positive organisms are relatively simple compounds, consisting of not more than 3 simple sugar amines, uronic acids, and they are usually antigenic without being particularly toxic. Also they are usually attached to cellular nucleo proteins. On the other hand gram-negative polysaccharides are more complex. They are composed of up to 6 monosaccharides, sugar amines and are firmly linked to a lipoid making a liposaccharide that is itself firmly attached to a lipoprotein. These compounds probably present in all gram-negative organisms have a very wide range of biological activity. They are highly toxic, pyrogenic, and cause disturbances to the blood cells such as leucopenia leucocytosis. They are all general metabolic stimulants, fibrinolytic, may prevent scar formation and can cause the necrotisation of tumours. Although some of these properties may find clinical application it must be remembered that the lipopolysaccharides are extremely toxic, and may cause, shortly after administration, a considerable lowering of the animal's resistance to other bacterial infections, before an increased resistance is subsequently established.

Workers from many countries have attempted the separation of these interesting compounds, but it is to Boivin and his colleagues (*e.g.* 1935, 1936) that the credit must go for being the first to demonstrate that polysaccharides may be isolated by simple methods. Boivin also termed each of his products the "antigene complet" because it was responsible for the total antigenicity, *e.g.* capsular and somatic, as well as for the other biological properties.

More recently Westphal and his school (1952a, b) have prepared highly purified polysaccharides and have subjected them to detailed chemical and biological examination. As the result of these brilliant researches many facts have emerged. Three of the most interesting are as quoted. First, the biological activity of the purified polysaccharides is very great, and small fractions of a microgram administered intravenously cause significant blood changes and pyrogenicity in man and other animals. Secondly, the polysaccharide fraction of the complex has been shown to possess, in addition to the simple sugars, glucose, mannose, galactose and rhamnose, members of a new family of hitherto undescribed sugars—the deoxymethylpentoses (*e.g.* Tyvelose, abequose; Davies *et al.*, 1958). These sugars are probably attached terminally in the polysaccharide and therefore confer a highly specific antigenicity. Thirdly, it has been shown that the lipoid fraction is responsible for most of the biological activity, causing effects previously mentioned. Although it is usually attached to polysaccharide it may be transferred to protein, when it still exercises its biological activity. Analysis of the lipoid reveals the presence of many well-known "units" such as lauric, myristic palmitic, β-hydroxy-myristic, phosphoric and aspartic acid, hexosamines, glycerol and one or more basic amines of which one of the most interesting is necrosine, a long chain of aliphatic diamine.

Little is known about the method or order of linking of these units, and there is no doubt that the exact elucidation of the structure of these lipoids will prove an important landmark in the field of bacterial chemistry.

While it cannot be questioned that these findings are of the utmost importance, it is not clear how they fit into the pattern of one of the most important features of gram negative activity, namely, enteritis. Gastroenteritis in babies often followed by pyelitis, and white scours in calves (Lovell 1937) are all too common and too serious and both are associated with, and probably caused by, pathogenic strains of the enterobacteriaceae.

In a recent study of the biochemical and chemical properties of 12 strains of *E. coli* of bovine origin Carne (1958) has shown that there are no obvious chemical or toxicological differences between Boivin type polysaccharides isolated from pathogenic and non-pathogenic sources. All were pyrogenic and toxic, and none could be induced to cause more than rare cases of scouring in small experimental animals. Thus, it seems that bacterial enteritis *per se* is associated primarily with the viability of organism, with the young host parasite relation and not wholly with the universal presence of highly toxic polysaccharides.

Clearly much remains to be done and the summarised problem appears to be to differentiate between pathogenic and non-pathogenic strains that all appear to possess nearly equal qualities of toxic lipopolysaccharides but do not all demonstrate the ability to scour the intestines of young animals.

The prime requirement is the development of more detailed and precise methods of biochemical and chemical analysis, preferably at molecular level in order that these important problems may be solved successfully.

D. G. Harvey, May, 1958.

References

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(*Concluded in col.* 1 *overleaf*)

Certificates of Heifers for Grading

AT a meeting between a representative of the Association and officials of the Fatstock Marketing Division of the Ministry of Agriculture, Fisheries and Food, the difficulties arising from the rejection by grading officers of veterinary certificates of non-pregnancy were fully discussed.

It was pointed out that the examination for pregnancy, as carried out by a veterinary surgeon, is a skilled and exact internal examination of the appropriate organs. This the veterinary surgeon is called upon to do frequently in the course of his daily routine. The appearance of the mammary gland, the body condition, or external palpation have no part in that examination, but on the contrary could be most misleading.

The B.V.A. representative was assured that the Ministry's officers will always take due note of properly completed veterinary certificates and of veterinary advice given on "suspect" animals. Where an officer is not prepared to accept such advice he will always seek the opinion of his superior officer, provided he is available. Where he still finds himself unable to certify the animal for the guarantee he will report to the producer of the animal in question and his veterinary adviser, if present, giving the reasons for rejecting the beast and advising that it be presented at a dead-weight centre where a fuller and more conclusive examination can take place.

At dead-weight centres, if the Fatstock Officer feels bound to reject the carcase, he will call in a superior officer for his opinion before doing so.

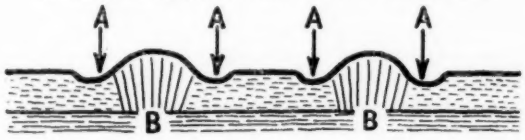
The regulations at present stipulate that the Grading Officers must make the final decision regarding eligibility of fatstock for guarantee. While it must be accepted, for the present, that there must be one person responsible for the final decision to pay the subsidy it will assist negotiations, should further difficulties arise, if certificates are always:—

1. Completed in duplicate. The certificate to accompany the animal to the grading centre and the duplicate to be retained by the veterinary surgeon.
2. Written on headed note-paper.
3. Provided with the date of examination.
4. So drawn up that the animal is clearly identified by ear number or ear tag.

At dead-weight centres the examination of the uterus by the following method will determine with accuracy whether an animal has ever been pregnant more than 3½ months.

1. Open each horn of the uterus and cut transversely through the wall including the diameter of a cotyledon.
2. Examine the blood vessels of the uterine wall and of the cotyledon.

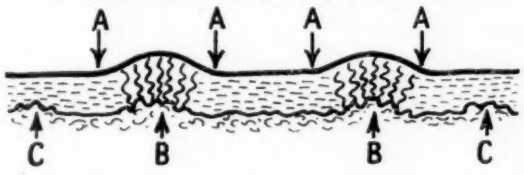
1.—CROSS SECTION OF WALL OF HEIFER'S UTERUS



A Shallow "moat" surrounding cotyledon.

B Blood vessels in cotyledon very fine and straight.

2.—CROSS SECTION OF WALL OF COW'S UTERUS



A. Absence of "moat."

B. Blood vessels which are distinct and contorted bulge from cut surface of cotyledon.

C. Blood vessels in wall very distinct and bulge.

Interpretation

(a) In the uterus of a heifer, the cotyledons are generally surrounded by a shallow "moat." In the cow, this "moat" disappears but this is not conclusive evidence.

(b) The blood vessels in the exposed wall of the uterus in the cow bulge from the cut surface and are contorted. In the heifer the blood vessels can be seen clearly but they do not bulge and show little contortion.

(c) The blood vessels in the cotyledons are the most reliable guide. In the heifer the blood vessels are very fine and straight. In the cow, the blood vessels are distinct and contorted and bulge slightly from the cut surface of the cotyledons.

The method assumes that the cotyledons must enlarge if the animal is in calf, and that they then regress in the non-pregnant uterus, *i.e.* that the animal must have been at least 3½ months in calf and possibly longer. This, it is suggested, is reasonable, since any abortion prior to this could have been missed by the owner.

There is another method which can be adopted, utilising X-ray examination in comparing the vascular supply of the virgin and post-gravid uterus, (*Brit. Vet. J.* **113**, (No. 12). 498).

In Brief—*Concluded.*

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News and Comment

RETIREMENT OF MR. C. W. FRANCIS

At a small party held on June 30th, the staff of the R.C.V.S. officially said "Farewell" to Mr. C. W. Francis, assistant secretary and accountant, and presented him with a clock. The official farewell by the Council of the Royal College will be made at the quarterly meeting in October next, when Mr. Francis will be formally presented with a television set, which was his choice of a parting gift.

More than three-quarters of the present members of the Royal College were admitted to membership, through examination arrangements made by Mr. C. W. Francis, during the period 1935 onwards when he was responsible for such arrangements. His service with the College dates from ten years earlier, when he was appointed, from 275 candidates, as assistant secretary in November, 1925. Before entering the service of the College, Mr. Francis had served for just over 3 years in the R.F.C., later to be better known as the R.A.F. The meticulous character of his work, which was later to be appreciated by the College, caused his release from the Forces to be delayed, for he served as a wireless operator with the Forces in Iraq. Between that period and his appointment to the College, he served in local government with the Hampstead Borough Council.

He was twice concerned with arrangements for International Veterinary Congresses. The first was in July, 1930, when he acted as assistant to Dr. Bullock, and his primary responsibilities were for the trades exhibition and the Congress banquet, in respect of which he received the special thanks of the organising committee. Many years later he was again responsible, in part, for the International Veterinary Congress of 1949 with all the difficulties of book-keeping in a period of restricted currencies.

Whilst Mr. Francis has always been associated with the accounts of the College, one of his principal tasks commenced in 1935—in which year he became a Fellow of the Chartered Institute of Secretaries—when he assumed responsibility for organising the M.R.C.V.S. and D.V.S.M. examinations. This always demanded considerable planning, with the visit of the panel to the various examination centres, but during the Second World War it was a matter of extreme difficulty, which he carried through to the satisfaction of all concerned. The task was made more difficult by the removal of the College headquarters from Red Lion Square to improvised headquarters at Harrogate, where Mr. and Mrs. Francis, as well as Dr. and Mrs. Bullock, worked long hours, with the minimum of staff, to keep all the arrangements going smoothly.

Shortly after the return to Red Lion Square the sudden and unexpected death of Dr. Bullock placed a great burden on Mr. Francis's shoulders, as the College was desperately short of staff. This was a particularly difficult period, owing to the discussions regarding the reports of the Loveday Committee. The appreciation of the Council was expressed to Mr. Francis, at this difficult period pending the appointment of a new Secretary and Registrar, by two resolutions of Council granting him special emoluments in recognition of his services. It was also at this period that he was granted the additional title of Accountant.

Although Mr. Francis is retiring from daily attendance at the College, he will still maintain a more active connexion with it than is often the case with retired members of the staff, for he will continue to act as a consultant on book-keeping matters, and in addition he will remain secretary of the Victoria Veterinary Benevolent Fund, in which he succeeded the late Dr. Bullock after a short interregnum, when the post was temporarily filled by the late Colonel Simpson. His many friends will be glad to feel that so old and valued a servant of the profession will still maintain regular contact with it, and everyone will wish him happiness and good health in his well-earned leisure.

VETERINARY COUNCIL OF IRELAND

At the biennial elections to fill three vacancies on the Veterinary Council of Ireland, Professor P. E. Mullaney, M.A., M.V.M., M.R.C.V.S., was elected a general elected member for four years; Mr. P. J. Daly, M.R.C.V.S., and Mr. A. D. Donnelly, M.R.C.V.S., were elected limited elected members for a similar period.

At a meeting of the Council, Professor J. A. Nicholson, M.A., M.V.SC., PH.D., M.R.C.V.S., was unanimously elected President of the council. Mr. J. A. Flynn, M.R.C.V.S., was unanimously elected hon. treasurer.

THE MILK INDUSTRY IN BARBADOS

The value to agricultural undertakings of expert, impartial advice from outside, is illustrated by the following extract from a leading article headed "Threat to Milk Industry" which appears in the *Barbados Advocate* of June 7th:—

"The situation is not a bright one and it is evident that something should be done about it before it is too late and the industry collapses and dies through economic strangulation. Fortunately a lead was given to the members of the two Associations by Mr. G. N. Gould, who was the guest speaker at the joint meeting. Mr. Gould is a veterinary surgeon and President of the Royal College of Veterinary Surgeons. He is on a tour of the area and spoke on the theme of farm management in Great Britain, the build-up of farm production under war and post-war conditions and the development of the veterinary profession during that period.

"In the course of his lecture, Mr. Gould advised the members of the Associations to organise the industry as had been done by the British farmer during the inter-war years when there was a depression in agriculture and it was thought that the depression would never end.

"He informed the meeting that he had discovered there was absolutely no system of marketing milk

in Barbados or any other products of livestock in the island. Without a system they could not have an industry, and he advised them to get together and do something to bring about a system of marketing the products of the industry.

"Anyone acquainted with the dairy and livestock industry in this island must appreciate the truth of Mr. Gould's observations."

NEMATODIRUS DISEASE

In Parliament, on June 16th, Mr. Whitelaw asked the Minister of Agriculture, Fisheries and Food, as representing the Lord President of the Council, what progress has been made in research into methods of preventing losses amongst lambs from nematodirus disease.

MR. JOHN HARE: A large amount of research bearing directly and indirectly on the problem of nematodirus is proceeding all over the world. Fundamental work on the life history and epidemiology of the parasite, which is necessary before any effective system of control can be introduced, is in progress at several agricultural research institutes in this country and work on finding new anthelminthic substances is also being carried on by the pharmaceutical industry. It is as yet too early to assess the effect of all this work in reducing losses from nematodirus.

MR. WHITELAW: May I, in the first place, apologise to my right hon. Friend for asking him a Question which necessitated his mentioning so many unpronounceable names in reply? May I call his attention to the fact that this nematodirus disease is spreading very fast in the north of England? Would it not be most valuable, therefore, if the highest priority were given to research so that effective methods of prevention may be found before the disease becomes a menace on a national scale?

MR. HARE: My hon. Friend is quite right. This is an important disease among hill sheep. I shall certainly see that the scale of research being devoted to it is adequate.

We understand, from correspondents, that this season's outbreaks of nematodirus in lambs is one of the most severe that has ever been experienced. Throughout the Border counties, in a large area north-east of Aberdeen, and in certain parts of central Wales a large proportion of the flocks have been seriously affected. We are advised that the experimental work using bephenium embonate* reported by Rawes and Scarnell in our issue of March 22nd, 1958, has been amply confirmed. The drug has been chiefly used in treatment, and even in seriously affected flocks improvement has been noticed as early as 48 hours after dosage. Its use for the control of the disease by routine dosage has not been so extensively applied, but where it has the lambs which have been dosed have remained healthy while the undosed controls have been badly affected, lost much weight, or died.

* "Frantin," Burroughs Wellcome & Company.

UNIVERSITY NEWS

Edinburgh

The undernoted candidates have passed the Part II Examination for the Diploma in Tropical Veterinary Medicine:—

Crosbie, Andrew, M.R.C.V.S.; Fadol, Gaafar Karrar, B.V.SC. (CAIRO); Hampshire, Frederick John William, M.R.C.V.S.; Kirby, Ian Ayrton Earle, D.V.M. & S. (TORONTO); Mares, Robert Gascoyne, M.R.C.V.S.; Nixon, Raymond Charles, B.V.SC. (PRETORIA); Shaib, Bukar, M.R.C.V.S.; Slater, Duthie, M.R.C.V.S.; and Thuraisingham, Sinnatamby, M.R.C.V.S.

The undermentioned candidates have passed the Third Professional Examination (Part I) in Veterinary Pharmacology for the degree of Bachelor of Veterinary Medicine and Surgery:—

Birch, Allen Vaughan; Dawson, Peter S.; Doxey, David L.; Eyre, Peter; Fairlie, George; Footman, Brian; Gill, Kenneth A. K.; Goad, Lewis; Harcourt, Ralph A.; Harrison, Richard D.; Irwin, Verney C. R.; Jackson, Peter G. G.; Mair, Malcolm L.; Middleton, Nigel T.; Moore, Alexander; Pratt, John H.; Radley, David E.; Reeves, Ronald J. C.; Robson, Richard ?; Rollo, David G.; Smith, Margaret E.; Somerville, John R.; Thomas, Gruffydd W.; Tindall, John R.; Wilkinson, Ian S.; and Williams, David A.

The undermentioned candidates have passed in the subject of Veterinary Anatomy and Histology of the Second Professional Examination for the B.V.M. & S. degree:—

Nicholson, Richard E.; Oduro, Gottfried A.; and Stewart, Hugh D.

The undermentioned candidates have passed in the subject of Veterinary Physiology (including Biochemistry) of the Second Professional Examination for the B.V.M. & S. degree:—

Abbott, John E. D.; Campbell, Edward G.; Duncan, Walter D.; Gardner, David W.; Goodman, Alan P.; Jobling, Terence; Jones, Meredydd; Lambert, Donald R.; Nicholson, Richard E.; Searles, Patricia A.; Taylor, Stuart M.; and Walton, John R.

The undermentioned candidate has passed the Third Professional Examination (Part II) in the subjects of Veterinary Pathology, Bacteriology, and Parasitology for the B.V.M. & S. degree:—

Hunt, George E.

The undermentioned candidates have passed in the subject of Veterinary Pathology of the Third Professional Examination (Part II) for the B.V.M. & S. degree:—

Atkinson, William I.; Logan, Ernest F.; Simpson, David T.; Wahler, Saw; and Watt, John G.

The undermentioned candidates have passed in the subject of Veterinary Bacteriology of the Third Professional Examination (Part II) for the B.V.M. & S. degree:—

Booth, James M.; and Gordon, Mary C. M.

The undermentioned candidate has passed in the subject of Veterinary Parasitology of the Third Professional Examination (Part II) for the B.V.M. & S. degree:—

James, William T.

Liverpool

ORDINARY DEGREE OF B.V.Sc.

Alderson, Valerie A.; Batt, R. A. L.; Beadle, G. G.; Bryan, N. M.; Davies, A.; Edwards, J.; Francis, P. G.; Hothersall, Maureen; Ingman, N. R.; Janes, P. D.; Lloyd, Anne S.;

Lloyd, R.; Llywelyn, G.; Macarthur, G. J.; Mears, Hazel; Pearson, P. D.; Perry, K. T. E.; Procter, G. M.; Sulley, M. J.; and Waterworth, J.

FINAL EXAMINATION (PART II) only

Barr, D. A.; Haywood, R. H.; McFarland, A. G.; and Penhale, W. J.

SECOND EXAMINATION

PART I (VETERINARY ANATOMY, INCLUDING HISTOLOGY AND EMBRYOLOGY)

Carding, A. H.; Green, P. B.; and Westbrook, R.

PART II (PHYSIOLOGY, INCLUDING BIOCHEMISTRY)

Jones, R. M.; Nairn, A. D.; Proudlove, A. J.; Thomson, Judith, A.; and Westbrook, R.

FIRST EXAMINATION

PART I (CHEMISTRY)

Bardgett, P. L.; Bradley, W. A.; Cameron, A.; Cope, B. T.; Fabry, A.; Gardner, W. B.; Gooderham, K. R.; Hall, Elizabeth; Hardy, Elizabeth A.; Harper, F. D. W.; Hilton, D. W.; Jones, R. T.; Jones, T. O.; Llewellyn, C. A.; Mohammed, I.; Moore, J. C.; Mudd, A. J.; Newcomb, R.; Pierrepoint, C. G.; Pulley, Norma M.; Rawson, B. C.; Riffkin, G. G.; Sopronfalvy, M.; Strong, M. G.; Summers, D.; Taylor, J. K.; Waud, R.; and Williams, H. G.

PART II (PHYSICS)

*Bardgett, P. L.; Bradley, W. A.; Cameron, A.; *Cope, B. T.; Curran, M. K.; Fabry, A.; Gardner, B.; Gooderham, K. R.; Hall, Elizabeth; Hallows, B. J.; Hammonds, Patricia; Hardy, Elizabeth A.; Harper, F. D. W.; Hilton, D. W.; Jones, R. T.; Jones, T. O.; *Llewellyn, C. A.; Mohammed, I.; Moore, J. C.; Mudd, A. J.; Newcomb, R.; *Pierrepoint, C. G.; Pulley, Norma M.; Rawson, B. C.; Riffkin, G. G.; Sopronfalvy, M.; Summers, D.; Taylor, J. K.; Thomas, I. B.; Thompson, Hannah S.; Waud, R.; and Williams, H. G.

PART III (BOTANY)

Bardgett, P. L.; Bradley, W. A.; Cameron, A.; Cope, B. T.; Curran, M. K.; Fabry, A.; Gardner, W. B.; Gooderham, K. R.; Hall, Elizabeth; Hallows, B. J.; Hammonds, Patricia; Hardy, Elizabeth A.; Harper, F. D. W.; Hilton, D. W.; Jones, R. T.; Jones, T. O.; Llewellyn, C. A.; Mohammed, I.; Moore, J. C.; Mudd, A. J.; Newcomb, R.; Pierrepoint, C. G.; Pulley, Norma M.; Rawson, B. C.; Riffkin, G. G.; Strong, M. G.; Summers, D.; Taylor, J. K.; Thomas, I. B.; Thompson, Hannah S.; Waud, R.; and Williams, H. G.

PART IV (ZOOLOGY)

Bardgett, P. L.; Bradley, W. A.; Cameron, A.; Cope, B. T.; Curran, M. K.; Fabry, A.; Gardner, W. B.; Gooderham, K. R.; Hall, Elizabeth; Hallows, B. J.; Hammonds, Patricia; *Hardy, Elizabeth A.; Harper, F. D. W.; Hilton, D. W.; Jones, R. T.; Jones, T. O.; Llewellyn, C. A.; Mohammed, I.; Moore, J. C.; Mudd, A. J.; Newcomb, R.; Pulley, Norma M.; Rawson, B. C.; Riffkin, G. G.; Rutherford, R.; Sopronfalvy, M.; Strong, M. G.; Summers, D.; Taylor, J. K.; Thomas, I. B.; Thompson, Hannah S.; Waud, R.; and Williams, H. G.

* Passed with distinction.

HOLIDAY EXCHANGE OF PRACTICES

A correspondent has written to make the suggestion that some veterinary surgeons might find it both pleasant and interesting to "exchange" practices for holiday periods in much the same way as clergymen exchange pulpits. Thus a member, say, in Devonshire might like to experience the workings of a colleague's practice in Lancashire, and the wives and families would benefit, it is thought, from the change of air and surroundings. Arrangements regarding the practice income in either case would, presumably, be a matter for friendly agreement between the parties.

It would be interesting to hear what readers think of this idea, and correspondence will be welcomed.

THE COLONIAL VETERINARY SERVICE

Promotions. H. Hardie, Veterinary Officer, Fiji, becomes senior veterinary officer (Poultry Laboratory), Northern Nigeria; J. E. Huddart, Veterinary Research Officer, becomes senior veterinary research officer, Kenya.

New appointments. R. M. Mackenzie, Veterinary Officer, British Honduras, and P. MacLeod, Veterinary Officer, Jamaica.

THE REGISTER OF VETERINARY SURGEONS

The undermentioned have been registered in the Commonwealth List of the Register of Veterinary Surgeons on the dates shown:—

FORSYTH, Bruce Adam, B.V.SC. (SYDNEY), c/o I.C.I. (A.N.Z.), Melbourne, Victoria, Australia, June 27th, 1958.

STEPHENS, Lindsay Ayton, B.V.SC. (SYDNEY), c/o Australia House, Strand, W.C.2, June 6th, 1958.

PERSONAL

Dr. E. Cotchin. The title of Reader in Veterinary Pathology has been conferred upon Dr. Cotchin in respect of his post at the Royal Veterinary College. Dr. Cotchin is, of course, honorary treasurer of the B.V.A., and the Association will be very happy in his academic advancement.

Births

FRENCH.—On June 7th, 1958, at Nairobi, Kenya, to Anne, wife of Peter J. French, M.R.C.V.S., a son, John James.

GREIG.—On March 21st, 1958, to Margaret Barclay (*née* Cooper), M.R.C.V.S., wife of William Alastair Greig, PH.D., B.SC., M.R.C.V.S., of 5, Jubilee Gardens, Bearsden, Glasgow, a daughter, a sister for Anne and Fiona.

HAXBY.—On June 27th, 1958, to Barbara, wife of D. L. Haxby, M.R.C.V.S., of Candant House, Upton, Newark, Notts, a son, Adrian John, brother for Joanna.

REES.—On June 25th, 1958, at 32, Greencroft, Mabelreign, S. Rhodesia, to Margaret (*née* Lovegrove) and Lynn, MS.R.C.V.S., a brother for Jennifer and Michael.

SWARBRICK.—On June 17th, 1958, to Ann, wife of Olaf Swarbrick, M.R.C.V.S., of Fontwell, Arundel, Sussex, a son.

R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

FORBES, William Gilmour, Carthgale, 21, Hill Street, Kilmarnock, Ayrshire. Graduated May 25th, 1901, at the Glasgow Veterinary School. Died January 15th, 1958.

GILBERT, Sydney Joseph, c/o Bank of N.S.W., Sydney, Australia, Major Australian Army Reserve. Graduated July 18th, 1912, at the Royal Veterinary College, London. Died April 14th, 1958.

WYLIE, Arthur Mill, The Dell, Grays, Essex. Graduated May 22nd, 1907, at the Edinburgh Veterinary School. Died June 24th, 1958, aged 73 years.

COMING EVENTS

July

9th (Wed.) Annual General Meeting of the Animal Diseases Research Association at the Moredun Institute, 3 p.m.

Summer Meeting of the South-East Midlands Veterinary Medical Association at the Cattle Breeding Centre, Little Horwood, Bucks, 5.45 p.m.

Summer Meeting of the A.V.T. and R.W.'s (Northern Region) at the Avoncroft Cattle Breeders, Stoke Prior, Bromsgrove, 12.15 p.m.

10th (Thurs.). Meeting of the Council of the Society of Practising Veterinary Surgeons at 7, Mansfield Street, Portland Place, 5 p.m.

Meeting of the South Wales Division in the Angel Hotel, Abergavenny, 7.30 p.m.

14th (Mon.) Meeting of the B.S.A.V.A. (Midland Region) at the Abbey Hotel, Malvern, Worcs., 7.30 p.m.

16th (Wed.) Meeting of the Scottish Metropolitan Division in the Royal (Dick) School of Veterinary Studies, Edinburgh, 4 p.m.

General Meeting of the Lancashire Veterinary Association at the University of Liverpool Veterinary Field Station, Leahurst, Neston, Wirral, 2.30 p.m.

Annual General Meeting of the Welsh Branch of the B.V.A. at the Belle Vue Hotel, Aberstwyth, 2.15 p.m.

17th (Thurs.). Meeting of the East Midlands Division at the King's Head Hotel, Loughborough, 7.30 p.m.

18th (Fri.) Summer Meeting of the Yorkshire Veterinary Society in the Royal Station Hotel, York, 2.30 p.m.

September

21st to 26th (Official Opening, Monday, September 22nd). 76th Annual Congress of the British Veterinary Association in Douglas, Isle of Man.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN LONDON

Wednesday, July 9th—No. 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Parliamentary and Public Relations Committee.
2.00 p.m. Finance Sub-committee.
3.45 p.m. Home Appointments Committee.

Thursday, July 10th—No. 7, Mansfield Street

9.30 a.m. Small-Animals Committee.
11.30 a.m. Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, July 11th—Connaught Rooms, Gt. Queen Street, W.C.2

10.15 a.m. Council.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Denbigh. Garthgynan Farm, Llanfair Dyffrin Clwyd, Ruthin (June 20).

Leics. Bondman Hayes Farm, Markfield Lane, Ratby (June 20).

Fowl Pest

Cambs. No. 6, Council Houses, Little Thetford, Ely (June 20).

Lancs. Wyngate House, Freckleton Road, Kirkham, Preston; Glenhurst, Chapel Lane, Longton, Preston (June 20).

Yorks. Grange Farm, Wincobank Lane, Sheffield, 4 (June 20).

Swine Fever

Berks. Bradford's Farm, Benham, Newbury (June 23).

Cambs. Staffurths Bridge Farm, Flood's Ferry, March Tubb's Farm, Sutton Gault, Sutton, Ely (June 20).

Ches. Home Farm, Victoria Road, Macclesfield (June 26).

Dorset. Park Farm, Martinstown, Dorchester (June 20); Henbury Farm Limited, Sturminster Marshall, Wimborne; Seymour's Farm, Wool, Wareham (June 23).

Essex. Reedlands, Weeley, Clacton-on-Sea (June 20).

Glouc.s. The Piggeries, 89, Manor Road, Fishponds, Bristol, 5 (June 23).

Herts. Highover Farm, Walsworth, Hitchin (June 20); Shenleybury Lane, Shenley, St. Albans (June 24).

Kent. Bardogs Farm, Toys Hill, Sevenoaks (June 20).

Lancs. Shepherd's Farm, Halsall Lane, Aughton, Ormskirk (June 23); Oak Farm, Oak Lane, Liverpool, 11 (June 25); The Grange, Lydgate, Oldham; Siddall Moor Farm, Hareshill Road, Heywood (June 26).

Leics. Milethorn Farm, Wolvey, Hinckley (June 24); 19 Holding, Land Settlement Association, Elmsthorpe, Earl Shilton; 21 Holding, Land Settlement Association, Elmsthorpe, Earl Shilton (June 26).

Middx. Hall Farm, Dennis Lane, Stanmore (June 23).

Norfolk. Denmark Green, Diss; Uphall Grange, Ashill, Thetford (June 20); White House Farm, Buxton Road, Horstead, Norwich (June 24); Bullocks Hill Farm, Wingfield, Diss (June 25); Dale Cottages, Thorpe Abbots, Diss (June 26).

Oxford University Farm, Bleddington (June 20).

Shrops. Ryefields, Whixhall, Whitchurch (June 20).

Somerset. Stembridge House, Stembridge, Kingsbury Episcopi, Martock; Little Island, Stembridge, Kingsbury Episcopi, Martock (June 25); Podimore Corner, Yeovilton, Yeovil; Gallers Plot, Yeovilton, Yeovil (June 26).

Staffs. The Piggeries at "Queslade," Queslett Road, Great Barr, Walsall; Avondale, Sheepcote Lane, Glascote, Tamworth (June 23); Woodhouse Farm, Bonningdale, Albrighton, Wolverhampton (June 25).

Suffolk. 53, The Street, Holywell Row, Bury St. Edmunds; Lodge Farm Cottage, Coney Weston, Bury St. Edmunds; Hall Farm, Freckenham, Bury St. Edmunds (June 23); Brittons Farm, Somerton, Bury St. Edmunds (June 25).

Sussex. Rickney Farm, Pevensey (June 20); Piggeries at Bishop's Field, Hoopers Lane, Malling Street, Lewes; Oakfields, Chalvington, Hailsham (June 23); Vue Valais, Northbridge Street, Robertsbridge; "Effingham," Albourne Road, Henfield (June 25).

Warwicks. Illshaw Heath Farm, Hockley Heath, Solihull (June 20); Oak Lodge, Husbands Bosworth, Rugby; Highfields Holdings, Husbands Bosworth, Rugby (June 25).

Yorks. Height Houses Farm, Walsden, Todmorden (June 24).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Clostridial Enteritis in Cattle

Sir,—Mr. Shirley's communication in THE VETERINARY RECORD of June 7th, recalls to me a series of cases I met in the Tenterden, Woodchurch, Wittersham area of the Romney Marsh in 1950 and 1951. The clinical picture was that of a cow down after calving and unable to rise. She would show some response to calcium borogluconate but later relapsed, developed a foul, blood-stained diarrhoea and died within 12 to 36 hours. One owner reported 2 cases which were heard to be groaning and found to be struggling violently and rolling: they both died within 30 minutes of the first symptoms being observed. *Post-mortem* examination of these and earlier cases showed enteritis, peritonitis and gas-gangrene of the abdominal musculature, but this gas-gangrene was sometimes much more generalised. The knacker's diagnosis was Romney Marsh "Struck." My diagnosis of Clostridial infection was not proved and the local bacteriologists suggested that the condition might be hypomagnesaemia.

After the first few cases a number were successfully treated as follows: A solution containing 4 oz. Ca. borogluconate and 1 oz. mag. sulphate was given half intravenously and half subcutaneously, 10 c.c. penicillin in oil (300,000 units per c.c.) intramuscularly and 100 c.c. pulpy kidney antiserum intravenously. The injections were repeated next day and smaller doses of penicillin only given for the next two or three days. The cow was usually normal by the fifth day. That these were not simple milk fever cases was shown by blood-stained diarrhoea which developed later on the first day but cleared up in about 24 hours. I found that penicillin and pulpy kidney antiserum had to be administered before the diarrhoea developed if the cow was to be saved. I was not able to save those cows which were treated as milk fever cases and were found to be scouring when a second visit was made, or those cows which had developed diarrhoea before any treatment was given.

Thus the successful treatment of these cases depended upon an early diagnosis. The cow appeared to be more intoxicated than the usual milk fever case, but that was the only obvious difference, the temperature was not elevated in my series of cases and was often sub-normal. I found the degree of response to an intravenous injection of Ca. borogluconate a useful guide: whereas the cow with milk fever would be almost normal within 15 minutes, the Clostridial enteritis cow would respond to the injection and then relapse again so that 15 minutes post-injection there would be no obvious improvement. Penicillin and pulpy kidney antiserum would then be given.

I have always felt that these cases were due to *Cl. welchii* type D but I do not think that there would be sufficient α antitoxin in pulpy kidney antiserum to influence an acute intoxication. In addition, the *post-mortem* picture that I encountered was much more generalised than that described by Mr. Shirley. It is possible that the penicillin was responsible for curing the condition, but I heard at the time that practitioners in the district using penicillin alone to treat apparently similar conditions had not been successful.

Fortunately, from my point of view as a general practitioner the cases suddenly ceased to appear. I had seen 9 or 10 in a period of about 7 or 8 months on different farms several miles apart, and I saw no more during the next 18 months. I then left the district so have no more recent information. I have subsequently met cases of acute hypomagnesaemia and the *post-mortem* picture is quite different.

I am interested by Mr. Shirley's remarks about a similar condition in pigs. During the past 15 years I have encountered a number of sudden deaths in pigs, usually sows, and in one case a boar, in which the *post-mortem* picture was that of acute generalised gas-gangrene. These cases were not anthrax, but no laboratory ever diagnosed the expected Clostridial infection.

Yours faithfully,
R. F. G. SANDERCOCK.

"The Paddocks,"
Frith Lane,
Mill Hill,
London, N.W.7.
June 10th, 1958.

The Isle of Man

Sir,—With reference to the article on the Isle of Man in your valuable journal dated May 10th, I regret very much to find that there was no mention in it of Sir Hall Caine who had very close connection with the Island and who rather endeared himself to sentimental overseas readers like myself, with his excellent novels based on customs and traditions of the natives of the "Isle."

Yours faithfully,
K. V. NATHAN.

Veterinary Department,
Raub, Pahang,
Malaya.
June 17th, 1958.

"Doctor" or "Mister"?

Sir,—Would it not be a retrograde step for your profession to ape the medical profession in wholesale annexation for your members of the title "Doctor," which strictly belongs to those who have taken a post-graduate degree?

Yours faithfully,
W. M. ALLCROFT.

Ministry of Agriculture, Fisheries and Food,
Great Westminster House,
Horseferry Road,
London, S.W.1.
June 19th, 1958.